

# PALM INTRANET

Day : Tuesday  
Date: 1/22/2008

Time: 09:59:20

## Inventor Name Search

Enter the **first few letters** of the Inventor's Last Name.  
Additionally, enter the **first few letters** of the Inventor's First name.

**Last Name**

**First Name**

To go back use Back button on your browser toolbar.

Back to [PALM](#) | [ASSIGNMENT](#) | [OASIS](#) | [Home page](#)

 **PALM INTRANET**Day : Tuesday  
Date: 1/22/2008

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Enter the **first few letters** of the Inventor's Last Name.

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**Last Name****First Name**

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## Refine Search

### Search Results -

Term	Documents
(5 NOT 6).PGPB,USPT,USOC,EPAB,JPAB,DWPI,TDBD.	55
(L5 NOT L6).PGPB,USPT,USOC,EPAB,JPAB,DWPI,TDBD.	55

**Database:**

US Pre-Grant Publication Full-Text Database  
 US Patents Full-Text Database  
 US OCR Full-Text Database  
 EPO Abstracts Database  
 JPO Abstracts Database  
 Derwent World Patents Index  
 IBM Technical Disclosure Bulletins

**Search:**

L7

### Search History

DATE: Tuesday, January 22, 2008 [Purge Queries](#) [Printable Copy](#) [Create Case](#)

<u>Set Name</u>	<u>Query</u>	<u>Hit Count</u>	<u>Set Name</u>
side by side			result set
<u>L7</u>	L5 not L6	55	<u>L7</u>
<u>L6</u>	L5 and (vector and (cancer or tumor))	98	<u>L6</u>
<u>L5</u>	L3 not L4	153	<u>L5</u>
<u>L4</u>	L3 and (bicistronic or multicistronic)	9	<u>L4</u>
<u>L3</u>	(p53 and (p14ARF or p19ARF))	162	<u>L3</u>
<u>L2</u>	L1 and (p53 and P14ARF)	2	<u>L2</u>
<u>L1</u>	Gjerset-Ruth-A\$.in.	7	<u>L1</u>

END OF SEARCH HISTORY

## Refine Search

### Search Results -

Term	Documents
CHEN-JIANDONG	4
CHEN-JIANDONGS	0
CHEN-JIANDONG.IN..PGPB,USPT,USOC,EPAB,JPAB,DWPI,TDBD.	4
(CHEN-JIANDONG.IN.).PGPB,USPT,USOC,EPAB,JPAB,DWPI,TDBD.	4

**Database:**  
 US Pre-Grant Publication Full-Text Database  
 US Patents Full-Text Database  
 US OCR Full-Text Database  
 EPO Abstracts Database  
 JPO Abstracts Database  
 Derwent World Patents Index  
 IBM Technical Disclosure Bulletins

**Search:**

### Search History

**DATE:** Tuesday, January 22, 2008    [Purge Queries](#)    [Printable Copy](#)    [Create Case](#)

<u>Set Name</u>	<u>Query</u>	<u>Hit Count</u>	<u>Set Name</u>
side by side			result set
DB=PGPB,USPT,USOC,EPAB,JPAB,DWPI,TDBD; THES=ASSIGNEE; PLUR=YES;			
OP=AND			
<a href="#">L5</a>	Chen-Jiandong.in.	4	<a href="#">L5</a>
<a href="#">L4</a>	L3 and (p14ARF)	0	<a href="#">L4</a>
<a href="#">L3</a>	Tanaka-Noriaki.in.	212	<a href="#">L3</a>
<a href="#">L2</a>	Tango-Yasuhisa.in.	0	<a href="#">L2</a>
<a href="#">L1</a>	Tiemann-Frank.in.	14	<a href="#">L1</a>

END OF SEARCH HISTORY

**Welcome to DialogClassic Web(tm)**

Dialog level 05.20.01D  
Last logoff: 14jan08 15:49:39  
Logon file1 22jan08 11:15:27

**\*\*\* ANNOUNCEMENTS \*\*\***  
\*\*\*

\*\*\*The 2008 EMTREE Thesaurus has been added to EMBASE (Files 72, 73, 772, and 972)

**NEW FILES RELEASED**

\*\*\*Trademarkscan - South Korea (File 655)

**RESUMED UPDATING**

\*\*\*File 154 & F155, MEDLINE

\*\*\*File 156, ToxFile

\*\*\*

**RELOADS COMPLETED**

\*\*\*Files 72 & 73, EMBASE

\*\*\*Files 340, 341 & 942, CLAIMS/U.S. Patents - 2006 reload now online

\*\*\*

**NEWS**

Chemical Structure Searching now available in Prous Science Drug Data Report (F452), Prous Science Drugs of the Future (F453), IMS R&D Focus (F445/955), Pharmaprojects (F128/928), Beilstein Facts (F390), Derwent Chemistry Resource (F355) and Index Chemicus (File 302). \*\*\*

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>>>and events, please visit What's New from Dialog at <<<  
>>><http://www.dialog.com/whatsnew/>. You can find news about<<<  
>>>a specific database by entering HELP NEWS <file number>.<<<  
>>>PROFILE is in a suspended state.  
>>>Contact Dialog Customer Services to re-activate it.

\* \* \*

File 1:ERIC 1965-2007/Nov  
(c) format only 2007 Dialog

Set Items Description

--- -----

Cost is in DialUnits

?

B 155, 159, 5, 73  
22jan08 11:15:49 User259876 Session D1065.1  
\$0.99 0.283 DialUnits File1  
\$0.99 Estimated cost File1  
\$0.10 INTERNET  
\$1.09 Estimated cost this search  
\$1.09 Estimated total session cost 0.283 DialUnits

SYSTEM:OS - DIALOG OneSearch

File 155: MEDLINE(R) 1950-2008/Jan 18  
(c) format only 2008 Dialog

\*File 155: MEDLINE has resumed updating. Please see HELP NEWS 154 for details.

File 159:Cancerlit 1975-2002/Oct  
(c) format only 2002 Dialog

File 5:Biosis Previews(R) 1926-2008/Jan W2  
(c) 2008 The Thomson Corporation

File 73:EMBASE 1974-2008/Jan 21

(c) 2008 Elsevier B.V.

\*File 73: The 2008 EMTREE Thesaurus has been loaded. Please see  
HELP NEWS 72 for details.

Set Items Description  
--- -----

?

S (P53 AND (P14ARF OR P19ARF))  
168322 P53  
2416 P14ARF  
715 P19ARF  
S1 1836 (P53 AND (P14ARF OR P19ARF))

?

S S1 AND (BICISTRONIC OR MULTICISTRONIC)  
1836 S1  
3742 BICISTRONIC  
212 MULTICISTRONIC  
S2 7 S1 AND (BICISTRONIC OR MULTICISTRONIC)

?

RD  
S3 5 RD (unique items)

?

T S2/3,K/ALL

2/3,K/1 (Item 1 from file: 155)  
DIALOG(R)File 155: MEDLINE(R)  
(c) format only 2008 Dialog. All rts. reserv.

14483839 PMID: 12957286

Tumor induction by an endogenous K-ras oncogene is highly dependent on  
cellular context.

Guerra Carmen; Mijimolle Nieves; Dhawahir Alma; Dubus Pierre; Barradas  
Marta; Serrano Manuel; Campuzano Victoria; Barbacid Mariano  
Molecular Oncology Programme, Centro Nacional de Investigaciones  
Oncologicas (CNIO), Melchor Fernandez Almagro 3, 28029, Madrid, Spain.

Cancer cell (United States) Aug 2003, 4 (2) p111-20, ISSN 1535-6108

--Print Journal Code: 101130617

Publishing Model Print

Document type: Journal Article; Research Support, Non-U.S. Gov't

Languages: ENGLISH

Main Citation Owner: NLM

Record type: MEDLINE; Completed

...a K-Ras(V12) oncprotein along with a marker protein (beta-geo) from a  
single bicistronic transcript. Expression of this oncogenic allele  
requires removal of a knocked in STOP transcriptional cassette...

...; akt; Reverse Transcriptase Polymerase Chain Reaction; Stem Cells  
--pathology--PA; Survival Rate; Tumor Suppressor Protein p14ARF --genetics  
--GE; Tumor Suppressor Protein p14ARF --metabolism--ME; Tumor Suppressor  
Protein p53 --genetics--GE; Tumor Suppressor Protein p53 --metabolism  
--ME

Chemical Name: Cdkn2a protein, mouse; Cyclin-Dependent Kinase Inhibitor  
p16; Proto-Oncogene Proteins; Tumor Suppressor Protein p14ARF ; Tumor  
Suppressor Protein p53 ; Protein-Serine-Threonine Kinases; Proto-Oncogene  
Proteins c-akt; Oncogene Protein p21(ras)

2/3,K/2 (Item 2 from file: 155)

DIALOG(R)File 155: MEDLINE(R)

(c) format only 2008 Dialog. All rts. reserv.

14387974 PMID: 12839954

**Enhanced tumor suppression by a p14ARF/p53 bicistronic adenovirus through increased p53 protein translation and stability.**

Huang Yinghui; Tyler Traci; Saadatmandi Neshat; Lee Casey; Borgstrom Per; Gjerset Ruth A

Sidney Kimmel Cancer Center, San Diego, California 92121, USA.

Cancer research (United States) Jul 1 2003, 63 (13) p3646-53, ISSN 0008-5472--Print Journal Code: 2984705R

Contract/Grant No.: CA69546; CA; NCI

Publishing Model Print; Erratum in Cancer Res. 2003 Aug 15;63(16) 5171

Document type: Journal Article; Research Support, Non-U.S. Gov't; Research Support, U.S. Gov't, Non-P.H.S.; Research Support, U.S. Gov't, P.H.S.

Languages: ENGLISH

Main Citation Owner: NLM

Record type: MEDLINE; Completed

**Enhanced tumor suppression by a p14ARF / p53 bicistronic adenovirus through increased p53 protein translation and stability.**

The p53 tumor suppressor controls a cell cycle arrest and apoptosis pathway that is central to tumor suppression and often disrupted in cancer. The accumulation and activity of p53 are positively controlled by the p14/ARF tumor suppressor and full restoration of the pathway in cancer cells may require that both p53 and p14ARF be supplied [corrected]. To address this issue, we have constructed a bicistronic adenoviral vector encoding the two proteins (Adp14/ p53) and compared its tumor suppressor activity with that of a single gene vector for p53 (Adp53). We find that tumor cells treated with Adp14/ p53 undergo a much sharper decrease in viability with increasing multiplicities of infection than do cells treated with Adp53, even when cells express endogenous p14ARF. Adp14/ p53 is also more effective than is a combination of single gene vectors for p14 and p53. The sharper decrease in cell viability after treatment of cells with Adp14/ p53 correlates with an increased rate of p53 protein synthesis and a decreased rate of p53 protein turnover, leading to increased steady-state levels of p53 protein and increased levels of p53 downstream targets mdm2, p21waf1, and bax. Adp14/ p53 treatment leads to an elevated bax:bcl2 ratio and induction of apoptosis in vitro and...

... failure of the tumor cells to induce neovascularization in vivo. The results indicate that endogenous p14ARF expression may be insufficient to ensure efficient accumulation of ectopic p53 after gene transfer and demonstrate that for tumor suppression, bicistronic coexpression of p14ARF and p53 is superior to p53 alone. The results show that in this setting, p14ARF promotes p53 accumulation by increasing p53 protein synthesis, in addition to its well-characterized ability to oppose mdm2-mediated degradation of p53.

...Descriptors: drug therapy--DT; \*Colonic Neoplasms--pathology--PA; \*Recombinant Fusion Proteins--toxicity--TO; \*Tumor Suppressor Protein p14ARF --genetics--GE; \*Tumor Suppressor Protein p53 --genetics--GE; \*Tumor Suppressor Protein p53 --metabolism--ME

Chemical Name: Antineoplastic Agents; Recombinant Fusion Proteins; Tumor Suppressor Protein p14ARF ; Tumor Suppressor Protein p53

2/3,K/3 (Item 1 from file: 5)

DIALOG(R)File 5:Biosis Previews(R)  
(c) 2008 The Thomson Corporation. All rts. reserv.

0019647937 BIOSIS NO.: 200700307678

**A bi-directionally controlled, modified p14ARF promoter linked to bicistronic p14 and t-BID induces selective apoptosis only in cancer cells with mutant ras and mutant p53.**

AUTHOR: Mao Yuehua (Reprint); Dinnen Richard D; Nichols Gwen; Brandt-Rauf Paul W; Fine Robert L

AUTHOR ADDRESS: Columbia Univ, New York, NY USA\*\*USA

JOURNAL: Proceedings of the American Association for Cancer Research Annual Meeting 48 p1170 APR 2007 2007

CONFERENCE/MEETING: 98th Annual Meeting of the American-Association-for-Cancer-Research Los Angeles, CA, USA April 14-18, 2007; 20070414

SPONSOR: Amer Assoc Canc Res

ISSN: 0197-016X

DOCUMENT TYPE: Meeting; Meeting Abstract

RECORD TYPE: Citation

LANGUAGE: English

**A bi-directionally controlled, modified p14ARF promoter linked to bicistronic p14 and t-BID induces selective apoptosis only in cancer cells with mutant ras and mutant p53 .**

DESCRIPTORS:

CHEMICALS & BIOCHEMICALS: ... p14ARF

GENE NAME: human p53 gene (Hominidae...)

**2/3,K/4 (Item 2 from file: 5)**

DIALOG(R)File 5:Biosis Previews(R)  
(c) 2008 The Thomson Corporation. All rts. reserv.

17542942 BIOSIS NO.: 200300497970

**Tumor suppression by a p14ARF/p53 bicistronic adenovirus.**

AUTHOR: Huang Yinghui (Reprint); Tyler Traci (Reprint); Saadatmandi Neshat (Reprint); Lee Casey (Reprint); Borgstrom Per (Reprint); Gjerset Ruth A (Reprint)

AUTHOR ADDRESS: Sidney Kimmel Cancer Center, San Diego, CA, USA\*\*USA

JOURNAL: Proceedings of the American Association for Cancer Research Annual Meeting 44 p660 July 2003 2003

MEDIUM: print

CONFERENCE/MEETING: 94th Annual Meeting of the American Association for Cancer Research Washington, DC, USA July 11-14, 2003; 20030711

ISSN: 0197-016X

DOCUMENT TYPE: Meeting; Meeting Abstract

RECORD TYPE: Citation

LANGUAGE: English

**Tumor suppression by a p14ARF / p53 bicistronic adenovirus.**

DESCRIPTORS:

...GENE NAME: bicistronic adenovirus-mediated tumor cell transfer, tumor growth suppressive effects...

... p53 tumor suppressor gene (Animalia...)

... bicistronic adenovirus-mediated tumor cell transfer, tumor growth suppressive effects

**2/3,K/5 (Item 3 from file: 5)**

DIALOG(R)File 5:Biosis Previews(R)  
(c) 2008 The Thomson Corporation. All rts. reserv.

17387493 BIOSIS NO.: 200300346212

**Enhanced tumor suppression by a p14ARF/p53 bicistronic adenovirus through increased p53 protein translation and stability.**

AUTHOR: Huang Yinghui; Tyler Traci; Saadatmandi Neshat; Lee Casey;  
Borgstrom Per; Gjerset Ruth A (Reprint)

AUTHOR ADDRESS: Sidney Kimmel Cancer Center, 10835 Altman Row, San Diego,  
CA, 92121, USA\*\*USA

AUTHOR E-MAIL ADDRESS: rgjerset@skcc.org

JOURNAL: Cancer Research 63 (13): p3646-3653 July 1, 2003 2003

MEDIUM: print

ISSN: 0008-5472 (ISSN print)

DOCUMENT TYPE: Article

RECORD TYPE: Abstract

LANGUAGE: English

**Enhanced tumor suppression by a p14ARF / p53 bicistronic adenovirus through increased p53 protein translation and stability.**

**ABSTRACT:** The p53 tumor suppressor controls a cell cycle arrest and apoptosis pathway that is central to tumor suppression and often disrupted in cancer. The accumulation and activity of p53 are positively controlled by the p14 ADP p14ARF ribosylation factor (AR) tumor suppressor, and full restoration of the pathway in cancer cells may require that both p53 and p14ARF be supplied. To address this issue, we have constructed a bicistronic adenoviral vector encoding the two proteins (Adp14/ p53) and compared its tumor suppressor activity with that of a single gene vector for p53 (Adp53). We find that tumor cells treated with Adp14/ p53 undergo a much sharper decrease in viability with increasing multiplicities of infection than do cells treated with Adp53, even when cells express endogenous p14ARF. Adp14/ p53 is also more effective than is a combination of single gene vectors for p14 and p53. The sharper decrease in cell viability after treatment of cells with Adp14/ p53 correlates with an increased rate of p53 protein synthesis and a decreased rate of p53 protein turnover, leading to increased steady-state levels of p53 protein and increased levels of p53 downstream targets mdm2, p21waf1, and bax. Adp14/ p53 treatment leads to an elevated bax:bcl2 ratio and induction of apoptosis in vitro and...

...failure of the tumor cells to induce neovascularization in vivo. The results indicate that endogenous p14ARF expression may be insufficient to ensure efficient accumulation of ectopic p53 after gene transfer and demonstrate that for tumor suppression, bicistronic coexpression of p14ARF and p53 is superior to p53 alone. The results show that in this setting, p14ARF promotes p53 accumulation by increasing p53 protein synthesis, in addition to its well-characterized ability to oppose mdm2-mediated degradation of p53.

**DESCRIPTORS:**

CHEMICALS & BIOCHEMICALS: p14ARF / p53 bicistronic adenovirus...

... p53 protein...

...stability, accumulation, translation, bicistronic coexpression...

...p14 ADP p14ARF ribosylation factor...

... p14ARF --...

... bicistronic coexpression

**2/3,K/6 (Item 1 from file: 73)**  
DIALOG(R)File 73:EMBASE  
(c) 2008 Elsevier B.V. All rts. reserv.

0079634700 EMBASE No: 2003342924

**Erratum: Enhanced tumor suppression by a p14ARF/p53 bicistronic adenovirus through increased p53 protein translation and stability (Cancer Research (July 1, 2003) (3646-3653))**

Huang Y.

Affiliation unspecified.

Cancer Research ( Cancer Res. ) (United States) August 15, 2003, 63/16 (5171)

CODEN: CNREA ISSN: 00085472

DOCUMENT TYPE: Journal; Erratum RECORD TYPE: Citation

LANGUAGE: English

**Erratum: Enhanced tumor suppression by a p14ARF / p53 bicistronic adenovirus through increased p53 protein translation and stability (Cancer Research (July 1, 2003) (3646-3653))**

**2/3,K/7 (Item 2 from file: 73)**  
DIALOG(R)File 73:EMBASE  
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0079558700 EMBASE No: 2003265558

**Enhanced tumor suppression by a p14ARF/p53 bicistronic adenovirus through increased p53 protein translation and stability**

Huang Y.; Tyler T.; Saadatmandi N.; Lee C.; Borgstrom P.; Gjerset R.A. // Gjerset R.A.

Sidney Kimmel Cancer Center, San Diego, CA 92121, United States // Sidney Kimmel Cancer Center, 10835 Altman Row, San Diego, CA 92121, United States.

AUTHOR EMAIL: rgjerset@skcc.org; rgjerset@skcc.org

CORRESP. AUTHOR: Gjerset R.A.

CORRESP. AUTHOR AFFIL: Sidney Kimmel Cancer Center, 10835 Altman Row, San Diego, CA 92121, United States

CORRESP. AUTHOR EMAIL: rgjerset@skcc.org

Cancer Research ( Cancer Res. ) (United States) July 1, 2003, 63/13 (3646-3653)

CODEN: CNREA ISSN: 00085472

DOCUMENT TYPE: Journal; Article RECORD TYPE: Abstract

LANGUAGE: English SUMMARY LANGUAGE: English

NUMBER OF REFERENCES: 45

**Enhanced tumor suppression by a p14ARF / p53 bicistronic adenovirus through increased p53 protein translation and stability**

The p53 tumor suppressor controls a cell cycle arrest and apoptosis pathway that is central to tumor suppression and often disrupted in cancer. The accumulation and activity of p53 are positively controlled by the p14 ADP p14ARF ribosylation factor (AR) tumor suppressor, and full restoration of the pathway in cancer cells may require that both p53 and p14ARF be supplied. To address this issue, we have constructed a bicistronic adenoviral vector encoding the two proteins (Adp14/ p53 ) and

compared its tumor suppressor activity with that of a single gene vector for p53 (Adp53). We find that tumor cells treated with Adp4/ p53 undergo a much sharper decrease in viability with increasing multiplicities of infection than do cells treated with Adp53, even when cells express endogenous p14ARF. Adp14/ p53 is also more effective than is a combination of single gene vectors for p14 and p53. The sharper decrease in cell viability after treatment of cells with Adp4/ p53 correlates with an increased rate of p53 protein synthesis and a decreased rate of p53 protein turnover, leading to increased steady-state levels of p53 protein and increased levels of p53 downstream targets mdm2, p21waf1, and bax. Adp14/ p53 treatment leads to an elevated bax: bc12 ratio and induction of apoptosis in vitro and...

...failure of the tumor cells to induce neovascularization in vivo, The results indicate that endogenous p14ARF expression may be insufficient to ensure efficient accumulation of ectopic p53 after gene transfer and demonstrate that for tumor suppression, bicistronic coexpression of p14ARF and p53 is superior to p53 alone. The results show that in this setting, p14ARF promotes p53 accumulation by increasing p53 protein synthesis, in addition to its well-characterized ability to oppose mdm2-mediated degradation of p53.

## DRUG DESCRIPTORS:

\*adenovirus vector; \*protein p14ARF --drug development--dv; \*protein p53 --drug development--dv

?

Set	Items	Description
S1	1836	(P53 AND (P14ARF OR P19ARF))
S2	7	S1 AND (BICISTRONIC OR MULTICISTRONIC)
S3	5	RD (unique items)

?

S S1 AND (VECTOR AND CANCER)  
1836 S1  
366294 VECTOR  
3058576 CANCER  
S4 41 S1 AND (VECTOR AND CANCER)

?

RD  
S5 29 RD (unique items)

?

S S5 NOT PY>2002.  
29 S5  
8807722 PY>2002  
S6 13 S5 NOT PY>2002

?

T S6/3,K/ALL

6/3,K/1 (Item 1 from file: 155)  
DIALOG(R)File 155: MEDLINE(R)  
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13924555 PMID: 12224024

Growth suppression by a p14(ARF) exon 1beta adenovirus in human tumor cell lines of varying p53 and Rb status.  
Saadatmandi Neshat; Tyler Traci; Huang Yinghui; Haghghi Ali; Frost Greg; Borgstrom Per; Gjerset Ruth A

Sidney Kimmel Cancer Center, San Diego, California 92121, USA.  
Cancer gene therapy (England) Oct 2002, 9 (10) p830-9, ISSN  
0929-1903--Print Journal Code: 9432230  
Contract/Grant No.: CA69546; CA; NCI  
Publishing Model Print  
Document type: Journal Article; Research Support, Non-U.S. Gov't;  
Research Support, U.S. Gov't, Non-P.H.S.; Research Support, U.S. Gov't,  
P.H.S.  
Languages: ENGLISH  
Main Citation Owner: NLM  
Record type: MEDLINE; Completed

**... suppression by a p14(ARF) exon 1beta adenovirus in human tumor cell lines of varying p53 and Rb status.**

We have analyzed the ability of an adenoviral vector encoding the exon 1beta region of the p14(ARF) tumor suppressor (ARF) to suppress the growth and viability of an array of tumor cell lines of various origins and varying p53 and Rb status, in order to establish the clinical potential of ARF. An important activity of ARF is regulation of p53 stability and function through binding to the mdm2 protein. By sequestering mdm2, ARF may promote...

... would be a strong candidate for therapeutic application, the possible dependence of ARF activity on p53 and Rb function presents a potential limitation to its application, as these functions are often impaired in cancer. We show here that a replication-defective adenovirus, Ad1beta, encoding the exon 1beta region of ARF is most effective in tumor cells expressing endogenous wild-type p53. Nevertheless, Ad1beta suppresses tumor cell growth and viability in vitro and in vivo, inducing G1...

... cell cycle arrest and cell death even in tumor cells lacking both functional Rb and p53 pathways, and independently of induction of the p53 downstream targets, p21, bax, and mdm2. These results point to an activity of ARF in human tumor cells that is independent of Rb or p53, and suggest that therapeutic applications based on ARF would have a broad clinical application in cancer.

Descriptors: \*Adenoviridae--genetics--GE; \*Neoplasms--pathology--PA; \*Nuclear Proteins; \*Retinoblastoma Protein--metabolism--ME; \*Tumor Suppressor Protein p14ARF --genetics--GE; \*Tumor Suppressor Protein p53 --metabolism--ME

...Chemical Name: Proteins; Proto-Oncogene Proteins; Proto-Oncogene Proteins c-bcl-2; Retinoblastoma Protein; Tumor Suppressor Protein p14ARF ; Tumor Suppressor Protein p53 ; bcl-2-Associated X Protein; Green Fluorescent Proteins; Trypan Blue; 5-bromo-4-chloro-3...

6/3,K/2 (Item 2 from file: 155)  
DIALOG(R)File 155: MEDLINE(R)  
(c) format only 2008 Dialog. All rts. reserv.

13873706 PMID: 12162819

**Adenovirus-mediated p14ARF gene transfer cooperates with Ad5CMV-p53 to induce apoptosis in human cancer cells.**

Tango Yasuhisa; Fujiwara Toshiyoshi; Itoshima Takahiro; Takata Yoshiko; Katsuda Kou; Uno Futoshi; Ohtani Shoichiro; Tani Tohru; Roth Jack A; Tanaka Noriaki

First Department of Surgery, Shiga University of Medical Science, Shiga 520-2192, Japan.

Human gene therapy (United States) Jul 20 2002, 13 (11) p1373-82,  
ISSN 1043-0342--Print Journal Code: 9008950

Contract/Grant No.: 2P50-CA70970-04; CA; NCI; CA16672; CA; NCI; P01  
CA78778-01A1; CA; NCI  
Publishing Model Print  
Document type: Comparative Study; Evaluation Studies; Journal Article;  
Research Support, Non-U.S. Gov't; Research Support, U.S. Gov't, P.H.S.  
Languages: ENGLISH  
Main Citation Owner: NLM  
Record type: MEDLINE; Completed

**Adenovirus-mediated p14ARF gene transfer cooperates with Ad5CMV- p53 to induce apoptosis in human cancer cells.**

p14(ARF), a product of the INK4A/ARF locus, induces p53 upregulation by neutralizing the effects of MDM2, a transcriptional target of p53 that antagonizes its function. Here we report that adenovirus-mediated p14(ARF) gene transfer leads to the accumulation of ectopically transduced p53 and to apoptosis in human cancer cells. We constructed an adenoviral vector expressing p14(ARF) (Ad-ARF) and examined its synergistic effect with p53-expressing adenovirus (Ad5CMV- p53 or Ad- p53) in human lung and esophageal cancer cells. Simultaneous Ad-ARF and Ad- p53 infection increased p53 protein levels not only in a wild-type p53-expressing cell line, but also in cell lines with deleted p53. This resulted in a significant in vitro cytotoxicity compared with Ad- p53 infection alone. Coinfection of Ad-ARF and Ad- p53 also resulted in an increase in expression of p53-inducible genes, including p21(WAF-1/Cip1), p53R2, and Noxa. In addition, the growth of human lung cancer tumors subcutaneously implanted into nu/nu mice was inhibited significantly by intratumoral injection with Ad-ARF and Ad- p53. Our data demonstrate that overexpression of ectopic p14(ARF) may render cells more sensitive to p53-mediated apoptosis, an outcome that has important implications for the treatment of human cancers.

Descriptors: \*Adenoviridae--genetics--GE; \*Apoptosis; \*Neoplasms, Experimental--therapy--TH; \*Tumor Suppressor Protein p14ARF --genetics--GE; \*Tumor Suppressor Protein p53 --genetics--GE...; Line; Cell Transformation, Viral; Esophageal Neoplasms--metabolism--ME; Esophageal Neoplasms--pathology--PA; Gene Expression; Genes, p53 --genetics--GE; Genetic Vectors; Humans; Lung Neoplasms--metabolism--ME; Lung Neoplasms --pathology--PA; Lung Neoplasms...

Chemical Name: Tumor Suppressor Protein p14ARF ; Tumor Suppressor Protein p53

**6/3,K/3 (Item 3 from file: 155)**

DIALOG(R)File 155: MEDLINE(R)  
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13811722 PMID: 12082630

**Adenovirus-mediated overexpression of p14(ARF) induces p53 and Bax-independent apoptosis.**

Hemmati Philipp G; Gillissen Bernhard; von Haefen Clarissa; Wendt Jana; Starck Lilian; Guner Dilek; Dorken Bernd; Daniel Peter T

Department of Hematology, Oncology and Tumor Immunology, Charite-Campus Berlin-Buch, Humboldt University, Lindenberger Weg 80, 13125 Berlin-Buch, Germany.

Oncogene (England) May 9 2002, 21 (20) p3149-61, ISSN 0950-9232--  
Print Journal Code: 8711562

Publishing Model Print

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: MEDLINE; Completed

**Adenovirus-mediated overexpression of p14(ARF) induces p53 and Bax-independent apoptosis.**

... unrelated tumor suppressor proteins, p16(INK4a) and p14(ARF), which are frequently inactivated in human cancer. Whereas p16(INK4a) acts through engagement of the Rb-cdk4/6-cyclin D pathway, both...

... functions of p14(ARF) were shown to be primarily dependent on the presence of functional p53. Recent reports have also implicated p14(ARF) in p53-independent mechanisms of cell cycle regulation and apoptosis induction, respectively. To further explore the pro-apoptotic function of p14(ARF) in relation to functional cellular p53, we constructed a replication-deficient adenoviral vector for overexpression of p14(ARF) (Ad-p14(ARF)). As expected, Ad-p14(ARF) efficiently induced apoptosis in p53/Rb wild-type U-2OS osteosarcoma cells at low multiplicities of infection. Interestingly, Ad-p14(ARF) also induced apoptosis in both p53-deleted SAOS-2 osteosarcoma cells and HCT116 colon cancer cells with a bi-allelic knock-out of p53 (HCT116-p53 (-/-)). Similarly, adenovirus-mediated overexpression of p14(ARF) induced apoptosis in p53/Bax-mutated DU145 prostate cancer cells as well as in HCT116 cells devoid of functional Bax (HCT116-Bax(-/-)). Restoration of...

... apoptotic DNA fragmentation irrespective of the presence or absence of either Bax or functional cellular p53. Nevertheless, overexpression of the anti-apoptotic Bcl-2 homolog Bcl-x(L) markedly inhibited p14...

... of Bax. Taken together, this report demonstrates the participation of signaling pathways apart from the p53/Mdm-2 rheostat and Bax in p14(ARF)-mediated apoptosis.

Descriptors: \*Apoptosis--physiology--PH; \*Proto-Oncogene Proteins --physiology--PH; \*Tumor Suppressor Protein p14ARF --physiology--PH; \*Tumor Suppressor Protein p53 --physiology--PH...; bcl-2--physiology--PH; Recombinant Fusion Proteins--physiology--PH; Tumor Cells, Cultured; Tumor Suppressor Protein p14ARF --genetics--GE; Tumor Suppressor Protein p53 --deficiency--DF; bcl-2-Associated X Protein; bcl-X Protein

...Chemical Name: Proto-Oncogene Proteins; Proto-Oncogene Proteins c-bcl-2; Recombinant Fusion Proteins; Tumor Suppressor Protein p14ARF; Tumor Suppressor Protein p53; bcl-2-Associated X Protein; bcl-X Protein

**6/3,K/4 (Item 4 from file: 155)**

DIALOG(R)File 155: MEDLINE(R)

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13467127 PMID: 11705881

**E2F-1 up-regulates c-Myc and p14(ARF) and induces apoptosis in colon cancer cells.**

Elliott M J; Dong Y B; Yang H; McMasters K M

Department of Surgery, James Graham Brown Cancer Center, University of Louisville, 529 South Jackson Street, Louisville, KY 40202, USA.

Clinical cancer research - an official journal of the American Association for Cancer Research (United States) Nov. 2001, 7 (11) p3590-7, ISSN 1078-0432--Print Journal Code: 9502500

Publishing Model Print

Document type: Journal Article; Research Support, Non-U.S. Gov't

Languages: ENGLISH

Main Citation Owner: NLM

Record type: MEDLINE; Completed

**E2F-1 up-regulates c-Myc and p14(ARF) and induces apoptosis in colon cancer cells.**

... In this study, we examine the ability of E2F-1 to induce apoptosis in colon cancer and the molecular mechanisms underlying E2F-1-mediated apoptosis. HT-29 and SW-620 colon adenocarcinoma cells (both mutant p53) were treated by mock infection or adenoviral vectors Ad5CMV (empty vector), Ad5CMVLacZ (beta-galactosidase), and Ad5CMVE2F-1 (E2F-1) at multiplicity of infection of 100. Western...

... Therefore, E2F-1 is a potentially active gene therapy agent for the treatment of colon cancer.

... Descriptors: Proteins; \*Proto-Oncogene Proteins c-myc--metabolism--ME; \*Transcription Factors--physiology--PH; \*Tumor Suppressor Protein p14ARF --metabolism--ME

... Chemical Name: Oncogene Proteins c-bcl-2; Proto-Oncogene Proteins c-myc; Transcription Factors; Tumor Suppressor Protein p14ARF

6/3,K/5 (Item 5 from file: 155)

DIALOG(R)File 155: MEDLINE(R)

(c) format only 2008 Dialog. All rts. reserv.

13042672 PMID: 11372376

[Increased radiosensitivity of lung cancer cell lines related with the functionally replaced p14ARF gene]

Gao N; Hu Y; Luo J  
Cancer Shanghai Institution, Medical University, Shanghai 200032, China.  
Zhonghua yi xue za zhi (China) Oct 2000, 80 (10) p776-9, ISSN  
0376-2491--Print Journal Code: 7511141

Publishing Model Print  
Document type: English Abstract; Journal Article; Research Support,  
Non-U.S. Gov't  
Languages: CHINESE  
Main Citation Owner: NLM  
Record type: MEDLINE; Completed

[Increased radiosensitivity of lung cancer cell lines related with the functionally replaced p14ARF gene]

OBJECTIVE: To assess the possibility that whether restored or enforced function of p14ARF could influence the radiosensitivity of lung cancer cells in vitro. METHODS: Human lung cancer cell lines with various endogenous backgrounds in INK4a, p53 and Rb genes were used as the recipients of the wild-type p14ARF gene. The expression of p14ARF mRNA and protein was detected with RT-PCR, immunohistochemistry and Western immunoblot after G418 selection. Clones expressing both p14ARF mRNA and protein were identified and selected for further experiments. By comparing with the parental and negative control cells prepared with empty vector, the effects of exogenously transfected p14ARF on cell cycle distribution, cell survival fraction and radiation-induced proportion of apoptosis were analyzed. RESULTS: The cell cycles of three wild-type p53 cell lines were arrested in G1 phase or G1 and G2-M phases. A significant decline in the proportion of S phase was observed in H460- p14ARF and A549- p14ARF cells, with decreased survival fraction and prolonged G2 delay after irradiation. We also observed in A549- p14ARF cells an increased percentage of apoptosis when radiated. CONCLUSION: The exogenously transfected wild-type p14ARF could increase the radiosensitivity of some lung cancer cells. It appears that cell cycle redistribution of cells after acquiring p14ARF may be the main explanation for the enhanced sensitivity. The increased apoptosis proportion of A549- p14ARF cells in response to radiation indicates a fortified p53 function and might partly contribute to the increased sensitization.

Descriptors: \*Lung Neoplasms--pathology--PA; \*Radiation Tolerance; \*Tumor

Suppressor Protein p14ARF --genetics--GE...; biosynthesis--BI; RNA, Messenger--genetics--GE; Tumor Cells, Cultured--radiation effects--RE; Tumor Suppressor Protein p14ARF --biosynthesis--BI  
Chemical Name: RNA, Messenger; Tumor Suppressor Protein p14ARF

6/3,K/6 (Item 6 from file: 155)

DIALOG(R)File 155: MEDLINE(R)

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12800580 PMID: 10914734

Induction of apoptosis in human esophageal cancer cells by sequential transfer of the wild-type p53 and E2F-1 genes: involvement of p53 accumulation via ARF-mediated MDM2 down-regulation.

Itoshima T; Fujiwara T; Waku T; Shao J; Kataoka M; Yarbrough W G; Liu T J ; Roth J A; Tanaka N; Kodama M

First Department of Surgery, Shiga University of Medical Science, Japan. Clinical cancer research - an official journal of the American Association for Cancer Research (UNITED STATES) Jul 2000, 6 (7) p2851-9, ISSN 1078-0432--Print Journal Code: 9502500

Publishing Model Print

Document type: Journal Article; Research Support, Non-U.S. Gov't

Languages: ENGLISH

Main Citation Owner: NLM

Record type: MEDLINE; Completed

Induction of apoptosis in human esophageal cancer cells by sequential transfer of the wild-type p53 and E2F-1 genes: involvement of p53 accumulation via ARF-mediated MDM2 down-regulation.

Transcriptional factor E2F-1 as well as tumor suppressor p53 have been shown to cause apoptosis independently in some types of human cancer cells when overexpressed. Here we report that sequential transfer of the wild-type p53 and E2F-1 genes efficiently induces apoptosis in human esophageal cancer cells and that E2F-1 overexpression directly, activates expression of p14 (ARF), which inhibits MDM2-mediated p53 degradation, resulting in the stabilization of p53. Infection of human esophageal cancer cell lines T.Tn and TE8 with adenovirus vector -expressing E2F-1 (Ad-E2F-1) enhanced mRNA and protein expression of ARF and decreased MDM2 protein expression. Transfection of ARF plasmid decreased MDM2 protein expression, which in turn increased p53 protein expression. Infection of T.Tn and TE8 cells first with adenovirus-expressing wild-type p53 (Ad-p53) and then with Ad-E2F-1 resulted in rapid induction of apoptosis; in contrast, simultaneous infection with Ad-E2F-1 and Ad-p53 had no significant antitumor effect. As shown by Western blot analysis, infection with suboptimal concentrations of Ad-E2F-1 induced the accumulation of exogenous p53 transduced by suboptimal concentrations of Ad-p53. Moreover, Ad-E2F-1-mediated ARF expression inhibited the up-regulation of MDM2 by overexpressed p53 in TE8 cells. Thus, overexpression of ectopic E2F-1 protein may stabilize endogenous as well as ectopic p53 protein via the E2F-1/ARF/MDM2/ p53 regulatory pathway and, in this way, render cells more sensitive to apoptosis, an outcome that...

...Descriptors: Cell Cycle Proteins; \*DNA-Binding Proteins; \*Esophageal Neoplasms--genetics--GE; \*Esophageal Neoplasms--pathology--PA; \*Genes, p53 ; \*Transcription Factors--genetics--GE; \*Transfection...; Recombinant Proteins--metabolism--ME; Transcription Factor DP1; Transcription, Genetic; Tumor Cells, Cultured; Tumor Suppressor Protein p14ARF ; Tumor Suppressor Protein p53 --genetics--GE

...Chemical Name: protein, human; Proteins; RNA, Messenger; Recombinant Proteins; Transcription Factor DP1; Transcription Factors; Tumor Suppressor Protein p14ARF ; Tumor Suppressor Protein p53 ; retinoblastoma binding

protein 1

6/3,K/7 (Item 7 from file: 155)  
DIALOG(R)File 155: MEDLINE(R)  
(c) format only 2008 Dialog. All rts. reserv.

12690874 PMID: 10778992

**Caspase activation and changes in Bcl-2 family member protein expression associated with E2F-1-mediated apoptosis in human esophageal cancer cells.**  
Yang H L; Dong Y B; Elliott M J; Liu T J; McMasters K M  
Department of Surgery, University of Louisville, James Graham Brown Cancer Center, Kentucky 40202, USA.

Clinical cancer research - an official journal of the American Association for Cancer Research (UNITED STATES) Apr 2000, 6 (4) p1579-89, ISSN 1078-0432--Print Journal Code: 9502500

Publishing Model Print

Document type: Journal Article; Research Support, Non-U.S. Gov't

Languages: ENGLISH

Main Citation Owner: NLM

Record type: MEDLINE; Completed

**...Bcl-2 family member protein expression associated with E2F-1-mediated apoptosis in human esophageal cancer cells.**

The prognosis for patients with esophageal cancer remains poor, prompting the search for new treatment strategies. Overexpression of E2F-1 has been shown to induce apoptosis in several cancer cell types. In the present study, the effect of adenovirus-mediated E2F-1 overexpression on human esophageal cancer cell lines Yes-4 and Yes-6 was evaluated. Cells were treated by mock infection, infection with an adenoviral vector expressing beta-galactosidase (Ad5CMV-LacZ), or E2F-1 (Ad5CMVE2F-1). Western blot analysis confirmed marked...

... revealed that overexpression of E2F-1 led to G2 arrest, followed by apoptotic cell death. p53 expression remained undetectable in both cell lines after E2F-1 overexpression. The apoptosis inhibitor proteins...

... at which apoptosis predominated, whereas pRb expression remained constant in Yes-6 cells. Expression of p14ARF did not change after E2F-1 infection in either cell line. Involvement of caspase 3...

... of the caspase 6 substrate, lamin B. These results indicate that the sensitivity of esophageal cancer cells to E2F-1-mediated apoptosis may be related to differential expression of Bcl-2...

...; biosynthesis--BI; Transcription Factor DP1; Transcription Factors --genetics--GE; Tumor Cells, Cultured; Tumor Suppressor Protein p53 --genetics--GE; bcl-X Protein

...Chemical Name: Oncogene Proteins c-bcl-2; Retinoblastoma Protein; Transcription Factor DP1; Transcription Factors; Tumor Suppressor Protein p53 ; bcl-X Protein; retinoblastoma binding protein 1; Caspases

6/3,K/8 (Item 8 from file: 155)  
DIALOG(R)File 155: MEDLINE(R)  
(c) format only 2008 Dialog. All rts. reserv.

12687895 PMID: 10772681

**Adenovirus-mediated p14(ARF) gene transfer in human mesothelioma cells.**  
Yang C T; You L; Yeh C C; Chang J W; Zhang F; McCormick F; Jablons D M  
Thoracic Oncology Laboratory, University of California, San Francisco

Cancer Center, CA 94115, USA.

Journal of the National Cancer Institute (UNITED STATES) Apr 19 2000,  
92 (8) p636-41, ISSN 0027-8874--Print Journal Code: 7503089  
Publishing Model Print  
Document type: Journal Article  
Languages: ENGLISH  
Main Citation Owner: NLM  
Record type: MEDLINE; Completed

... locus promotes degradation of the MDM2 protein and thus prevents the MDM2-mediated inhibition of p53. Homozygous deletion of the INK4a/ARF locus is common in human mesothelioma and may result in the loss of p14(ARF) and the inactivation of p53. We designed this study to evaluate the biologic and potential therapeutic roles of p14(ARF) expression in mesothelioma cells. Methods and Results: We constructed Adp14, an adenoviral vector carrying human p14(ARF) complementary DNA, and used it to transfect human mesothelioma cell lines H28, H513, H2052, and MSTO-211H. Overexpression of p14(ARF) led to increased amounts of p53 and the p21(WAF) proteins and dephosphorylation of the retinoblastoma protein. The growth rate of...

... markedly by infection with Adp14 compared with mock infection or infection with a control adenovirus vector, AdCtrl. Overexpression of p14(ARF) induced G(1)-phase cell cycle arrest and apoptotic cell...

... Cytotoxicity assays showed that Adp14 had a statistically significantly (P = .002) greater effect on colon cancer (HCT116) cell lines containing two copies of the wild-type p53 gene than on p53 -null cells, suggesting that functional p53 is a critical determinant of p14(ARF)-mediated cytotoxicity. CONCLUSIONS: The transfection of p14(ARF...

...; Cyclins--metabolism--ME; Genetic Vectors; Humans; Mesothelioma --metabolism--ME; Tumor Cells, Cultured; Tumor Suppressor Protein p14ARF ; Tumor Suppressor Protein p53 --metabolism--ME

Chemical Name: CDKN1A protein, human; Cyclin-Dependent Kinase Inhibitor p21; Cyclins; Proteins; Tumor Suppressor Protein p14ARF ; Tumor Suppressor Protein p53

6/3,K/9 (Item 1 from file: 5)

DIALOG(R)File 5:Biosis Previews(R)

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16923884 BIOSIS NO.: 200200517395

**Role for the double-stranded RNA activated protein kinase PKR in E2F-1-induced apoptosis**

AUTHOR: Vorburger Stephan A; Pataer Abujiang; Yoshida Kazumi; Barber Glen N ; Xia Weiya; Chiao Paul; Ellis Lee M; Hung Mien-Chie; Swisher Stephen G; Hunt Kelly K (Reprint)

AUTHOR ADDRESS: Department of Surgical Oncology, University of Texas M.D. Anderson Cancer Center, 1515 Holcombe Blvd, Box 444, Houston, TX, 77030-4009, USA\*\*USA

JOURNAL: Oncogene 21 (41): p6278-6288 12 September, 2002 2002

MEDIUM: print

ISSN: 0950-9232

DOCUMENT TYPE: Article

RECORD TYPE: Abstract

LANGUAGE: English

...ABSTRACT: of E2F-1 provokes apoptosis in a wide variety of malignant cells. To date only p14ARF and p73, a p53 homologue, have been

identified as E2F-1-inducible genes capable of mediating an apoptotic response. Here we show that adenovirus-mediated E2F-1 overexpression in cancer cells induces expression and autophosphorylation of the double-stranded RNA-dependent protein kinase PKR leading...

...and to apoptotic cell death. This PKR-dependent apoptosis occurs in cell lines with mutated p53 and in cell lines with mutated p53 and p73, and is significantly reduced by the chemical inhibition of PKR activation. Further, PKR...

...pathway of E2F-1-mediated apoptosis is dependent on PKR activation and does not require p53 or p73.

DESCRIPTORS:

...ORGANISMS: gene vector ; ...

...human breast cancer cells...

...human breast cancer cells

...GENE NAME: human p53 gene (Hominidae...)

6/3,K/10 (Item 2 from file: 5)

DIALOG(R)File 5:Biosis Previews(R)

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16826134 BIOSIS NO.: 200200419645

**Adenovirus-mediated p14ARF gene transfer cooperates with Ad5CMV-p53 (INGN 201) to induce apoptosis in human cancer cells**

AUTHOR: Tango Yasuhisa (Reprint); Fujiwara Toshiyoshi; Kataoka Masafumi; Kagawa Shunsuke; Ohtani Shoichiro; Tsunemitsu Yosuke; Tokunaga Naoyuki; Tani Tohru; Tanaka Noriaki

AUTHOR ADDRESS: Shiga University of Medical Science, Otsu, Japan\*\*Japan

JOURNAL: Proceedings of the American Association for Cancer Research Annual Meeting 43 p1100 March, 2002 2002

MEDIUM: print

CONFERENCE/MEETING: 93rd Annual Meeting of the American Association for Cancer Research San Francisco, California, USA April 06-10, 2002;

20020406

ISSN: 0197-016X

DOCUMENT TYPE: Meeting; Meeting Abstract

RECORD TYPE: Citation

LANGUAGE: English

**Adenovirus-mediated p14ARF gene transfer cooperates with Ad5CMV-p53 (INGN 201) to induce apoptosis in human cancer cells**

DESCRIPTORS:

...ORGANISMS: gene vector ; ...

...apoptosis, human lung cancer cells...

...apoptosis, human lung cancer cells...

...apoptosis, human lung cancer cells

DISEASES: lung cancer --

...GENE NAME: human p14ARF gene (Hominidae...)

...human p53 gene (Hominidae...)

6/3,K/11 (Item 3 from file: 5)

DIALOG(R)File 5:Biosis Previews(R)  
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16774044 BIOSIS NO.: 200200367555

**Anti-tumor activities of p14ARF in breast and prostate cancer**

AUTHOR: Huang Yinghui (Reprint); Saadatmandi Neshat (Reprint); Tyler Traci (Reprint); Haghghi Ali (Reprint); Frost Greg (Reprint); Borgstrom Per (Reprint); Gjerset Ruth A (Reprint)

AUTHOR ADDRESS: Sidney Kimmel Cancer Center, San Diego, CA, USA\*\*USA

JOURNAL: Proceedings of the American Association for Cancer Research Annual Meeting 43 p91 March, 2002 2002

MEDIUM: print

CONFERENCE/MEETING: 93rd Annual Meeting of the American Association for Cancer Research San Francisco, California, USA April 06-10, 2002; 20020406

ISSN: 0197-016X

DOCUMENT TYPE: Meeting; Meeting Abstract

RECORD TYPE: Citation

LANGUAGE: English

**Anti-tumor activities of p14ARF in breast and prostate cancer**

DESCRIPTORS:

...ORGANISMS: gene vector ; ...

...gene vector

DISEASES: breast cancer --...

...prostate cancer --

CHEMICALS & BIOCHEMICALS: p14ARF --...

... p53 --

**6/3,K/12 (Item 4 from file: 5)**

DIALOG(R)File 5:Biosis Previews(R)

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16141358 BIOSIS NO.: 200100313197

**The human tumor suppressor ARF interacts with spinophilin/neurabin II, a type 1 protein-phosphatase-binding protein**

AUTHOR: Vivo Maria; Calogero Raffaele A; Sansone Federica; Calabro Viola; Parisi Tiziana; Borrelli Loredana; Saviozzi Silvia; La Mantia Girolama (Reprint)

AUTHOR ADDRESS: Department of Genetics, General and Molecular Biology, University of Naples "Federico II", Via Mezzocannone 8, Napoli, 80134, Italy\*\*Italy

JOURNAL: Journal of Biological Chemistry 276 (17): p14161-14169 April 27, 2001 2001

MEDIUM: print

ISSN: 0021-9258

DOCUMENT TYPE: Article

RECORD TYPE: Abstract

LANGUAGE: English

ABSTRACT: The INK4a gene, one of the most often disrupted loci in human cancer, encodes two unrelated proteins, p16INK4a and p14ARF (ARF) both capable of inducing cell cycle arrest. Although it has been clearly demonstrated that ARF inhibits cell cycle via p53 stabilization, very little is known about the involvement of ARF in other cell cycle regulatory...

...formation of G418-resistant colonies when transfected into human and mouse cell lines, regardless of p53 and ARF status. Moreover, spinophilin/ARF coexpression in Saos-2 cells, where ARF ectopic expression...

**DESCRIPTORS:**

CHEMICALS & BIOCHEMICALS: ... p53 ;

...METHODS & EQUIPMENT: gene expression/ vector techniques, genetic method

6/3, K/13 (Item 1 from file: 73)

DIALOG(R)File 73:EMBASE

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0079392144 EMBASE No: 2003096093

**Selectively replicating adenoviruses for cancer therapy: An update on clinical development**

Post L.E.

Onyx Pharmaceuticals Inc., 3031 Research Drive, Richmond, CA 94806, United States

AUTHOR EMAIL: lpost@onyx-pharm.com

CORRESP. AUTHOR: Post L.E.

CORRESP. AUTHOR AFFIL: Onyx Pharmaceuticals Inc., 3031 Research Drive, Richmond, CA 94806, United States

CORRESP. AUTHOR EMAIL: lpost@onyx-pharm.com

Current Opinion in Investigational Drugs ( Curr. Opin. Invest. Drugs' ) ( United Kingdom) December 1, 2002, 3/12 (1768-1772)

CODEN: CIDRE ISSN: 14724472

DOCUMENT TYPE: Journal; Review RECORD TYPE: Abstract

LANGUAGE: English SUMMARY LANGUAGE: English

NUMBER OF REFERENCES: 49

**Selectively replicating adenoviruses for cancer therapy: An update on clinical development**

...015 (CI-1042, dl1520; Onyx Pharmaceuticals Inc), which replicates selectively in cells deficient in the p53 pathway, was the first such adenovirus to reach clinical testing. Multiple trials of ONYX-015 in over 300 cancer patients, and trials with other selectively replicating adenoviruses, have established the safety of this approach...

...other approaches, provide promising directions to develop selectively replicating adenoviruses into systemic therapy for metastatic cancer .

**DRUG DESCRIPTORS:**

adenovirus vector ; alpha fetoprotein--endogenous compound--ec; antineoplastic agent--adverse drug reaction--ae; antineoplastic agent --clinical trial...

...endogenous compound--ec; prostate specific antigen--endogenous compound --ec; protein MDM2--endogenous compound--ec; protein p14ARF --endogenous compound--ec; protein p53 --endogenous compound--ec; thymidine kinase --endogenous compound--ec; transcription factor E2F--endogenous compound --ec; tumor...

**MEDICAL DESCRIPTORS:**

\* cancer --drug therapy--dt; \*virus replication  
abdominal pain--side effect--si; Adenovirus; antineoplastic activity;  
cancer combination chemotherapy; cancer radiotherapy; cancer survival;  
chill--side effect--si; clinical trial; colorectal cancer --drug therapy  
--dt; Cytomegalovirus; diarrhea--side effect--si; drug distribution; drug

efficacy; drug fever--side...

...toxicity--side effect--si; enzyme activation; gene expression; gene insertion; gene mutation; head and neck cancer --drug therapy--dt; hepatic artery; human; liver cell carcinoma--drug therapy--dt; liver dysfunction --side...

...metastasis--drug therapy--dt; low drug dose; metastasis--drug therapy --dt; monotherapy; mouthwash; nonhuman; ovary cancer --drug therapy--dt; prostate cancer --drug therapy--dt; prostate cancer --radiotherapy--rt; protein defect; recurrent cancer --drug therapy--dt; recurrent cancer --radiotherapy--rt; review; rigor--side effect--si; side effect--side effect--si; systemic therapy; tooth...

SECTION HEADINGS:

...Virology

Adverse Reactions Titles

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Clinical and Experimental Pharmacology

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S1	1836	(P53 AND (P14ARF OR P19ARF))
S2	7	S1 AND (BICISTRONIC OR MULTICISTRONIC)
S3	5	RD (unique items)
S4	41	S1 AND (VECTOR AND CANCER)
S5	29	RD (unique items)
S6	13	S5 NOT PY>2002

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\$42.70 Estimated cost this search  
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